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1. Introduction

Malassezia dermatitis is common in dogs, and less common in cats.[1,2] *Malassezia* spp. are commensal yeasts that usually colonize the superficial epidermis of the skin and the ears. The non-lipid dependend M. *pachydermatis* and the lipid dependent M. *sympodialis*, M. *globosa, M. nana, M. slooffiae, M. restricta* and M. *furfur* are usually found on the skin or mucosae of dogs and cats.[1-3] Because *Malassezia* sp. do not invade subcorneally, it is hypothesized that the dermatitis results from inflammatory and/or hypersensitivity reactions to yeast enzymatic products and antigens.[4] *Malassezia*-specific IgG and IgE seem to be higher in atopic then normal dogs pointing toward a possible involvement in the pathogenesis of atopic dermatitis.[5,6] This was also shown in humans.[7]

Predisposing or triggering factors for *Malassezia* dermatitis development are as follows: increased humidity, presence of skin folds, endocrinopathies (hypothyroidism, diabetes mellitus, hyperadrenocorticism), increased populations of staphylococci, immunologic alterations (allergies, FIV, neoplasia, glucocorticoid therapy), and genetic predisposition (Basset hound, West Highland White Terrier, American Cocker Spaniel, and many other dog breeds, short-hair cat breeds such as Sphynx and Rex cats).[1-3,8-12]

2. Clinical signs

Predominant skin lesion in dogs are erythema, scaling, greasiness, crusts, in chronic cases also lichenification and hyperpigmentation (localized or generalized).[2] Sometimes the disease is associated with papules and even furunculosis.[2] If affected, the claws show red-brown discoloration. Predilection site are lips, ear canals, groin, ventral neck, medial thights, perianal area, interdigital skin and skin folds. In addition to skin changes, nearly all patients experience pruritus.[2] In the cat, *Malassezia* dermatitis should be on the differential list, in following clinical presentations:

- Otitis externa (often with accumulations of brown/black and waxy material)
- Chin acne
- Facial dermatitis
- Paronychia and/or red-brown discoloration of the claws
- Generalized erythematous scaly to waxy dermatitis[2,8,11,13,14]

3. Diagnosis

Main differential diagnoses include allergic dermatitis, contact dermatitis, superficial staphylococcal folliculitis, demodicosis, dermatophytosis, scabies, cheyletiellosis, feline acne and epitheliotropic lymphoma. Remember that most of the dogs and cats with *Malassezia* dermatitis have concurrent dermatoses or a systemic disease, which should be screened for.

The diagnosis is made by cytological examination using direct impressions with a microscopic slide, swab, superficial scraping or cellophane tape strippings (for less accessible areas such as interdigitial skin or dry lesions).[2] Whichever method is used, the glass slides are usually stained with Romanowsky stain (Diff-Quik®). Optionally, the samples (not scotch tape) can be heat

fixated before staining.[15] This may prevent loss of some material and consequently important information during the staining procedure. If one is searching only infectious agents such as bacteria and *Malassezia* just the basophilic stainining component may be used. Additional utilization of the eosinophilic stain reveals important information about the inflammatory response (Example eosinophils). The yeasts are best visualized at 40x or 100x magnification and are round to oval shaped with or without budding (classical "peanut shape"). The yeast diameter maybe from 3-8 um in diameter.[2] There is no definite yeast number to diagnose *Malassezia* dermatitis. Therefore, the diagnostic criteria for *Malassezia* dermatitis should always be interpreted in the clinical context. Some cases even require a therapeutical trial, to confirm the diagnosis. The fungal culture is of **no diagnostic value** in superficial infections, as dogs and cats can be healthy carriers. Important is to have typical clinical lesions with associated *Malassezia* organisms found on the cytological examination.

4. Treatment

Treatment options depend on the severity of lesions and the compliance of the owner/patient. Small focal areas can be treated with antifungal creams, wipes or ear drops. In multifocal or even generalized disease the topical therapy includes application of shampoos and rinses in mono- or combined preparations: chlorhexidin, ketoconazole, miconazole, climbazole, selenium sulfide, enilconazole, lime sulfur, acetid and boric acid. When topical therapy is impractical or ineffective, oral antifugal treatment may be instituted. The most commonly used drugs are itraconazole, ketoconazole, terbinafine. [13,16-21] As the therapeutic levels of systemic antifungals persist in the skin for several days or weeks, pulse regimens are possible. Newer treatment modalities, such as killer peptides, are being described recently.[22] Very important is to consider possible drug interactions when creating a multimodal therapeutic regimen (example allergic dogs treated with cyclosporine). As infections with *Malassezia* sp. often co-exist with staphylococcal infections, do not forget to diagnose and treat also these. Patients with a clinically relevant inflammatory component (usually allergic dogs), require additional anti-inflammatory drugs, such as glucocorticoids. Antifungal susceptibility testing is not routinely performed, but should be considered in refractory cases, as azole-resistant cases have been already encountered in veterinary medicine.[23]

Dramatic clinical improvement is seen within 1 to 2 weeks (faster if topical and systemic medications are combined). The duration of the therapy depends on the resolution of clinical signs and should be continued 7-10 days beyond clinical cure, in average altogether 4 weeks.[2]

Recurrent cases of *Malassezia* dermatitis are not unusual, as generally an underlying chronic disease is present. Frequent recurrences may require a maintenance protocol with utilization of either regular topical (once to twice weekly) or systemic antifungals (1-3 days/week pulses).

Again, to achieve a good clinical response to treatment, all efforts should be made to identify and correct the predisposing factors (allergy, hormonal disturbances, neoplasia, immune-deficiencies, etc).

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